EXHIBIT C

KENNEDY HODGES L.L.P

TRIAL ATTORNEYS

Bair Hugger Warming and Peri-Prosthetic Infections in Joint Replacement Surgery:

A Guide to Product Liability Litigation.

Table of Contents

	Page
Forward	
Note from David Hodges, Esq.	**3**
Abstract	**4**
Tommy Walton v. 3M et al.	**6**
Periprosthetic joint infections ("PJIs") compared to surgical site infections ("SSIs")	**8**
How does Bair Hugger's waste heat cause PJIs?	**11**
Bair Hugger's internal contamination: an additional risk	**17**
Chain of infection analysis	**21**
The McGovern study: critique and rebuttal	**27**
3M's knowledge of the risks of Bair Hugger	**29**
3M's improper actions and misrepresentations	**32**
Timeline of events	**36**
3M/Arizant key players	**40**
Dr. Daniel Sessler	**41**
Legal documents	
Detailed complaint	**46**
Kennedy Hodges Petition	**46**
Document Requests	**46**

Forward:

A note from David W. Hodges, Esq.,

Kennedy Hodges assembled the information in this Guide over the last two-plus years. The vast majority of underlying data comes from public documents: studies published in peer-reviewed medical journals, marketing materials distributed publicly by 3M/Arizant or its competitors, and documents available from various websites.

As the Guide explains, the inventor of Bair Hugger forced-air warming discovered the dangerous unintended consequences of his invention while developing an alternative warming methodology. He apparently notified 3M/Arizant of these flaws on multiple occasions. Despite these warnings, 3M/Arizant has not alerted clinicians of the risk, has not relabeled the product with warnings or contraindications for use and, most importantly, has not recalled or fixed the problems.

There are 12,000-20,000 orthopedic implant infections in the United States each year. Bair Hugger warming was used in a majority of those.

We believe that Mr. Walton's case is the first of hundreds or even thousands of Bair Hugger product liability cases that may eventually be filed against 3M. We are taking a leadership role in this effort to win well-deserved compensation for these horribly injured and permanently disabled patients by sharing our "road map" of the evidence for these cases with you.

We hope this encourages you and other lawyers to market for these cases. Our firm of seven attorneys stands ready to assist you as attorney in charge, co-counsel, or any other role that you feel is appropriate as this litigation proceeds. We welcome your referrals.

Please feel free to call us if you have questions regarding this material.

David W. Hodges, Esq.
Partner, Kennedy Hodges LLP,
711 West Alabama Street
Houston, TX 77006
(713) 523-0001
www.kennedyhodges.com

Abstract:

Background

For decades, plaintiffs' personal injury lawyers have rejected surgical infection cases. No matter how horrific the injury, how sympathetic the plaintiff, or how huge the damages, the injured person was left without recourse. Proving causation was simply too difficult. For infections following joint replacement surgery, at least, that obstacle has nearly vanished.

Research published over the past 18 months shows that, in most cases, there is an identifiable likely cause: Bair Hugger forced-air patient warming systems.

Bair Hugger operates like a hair dryer—taking ambient air from the operating room and heating it to approximately 117° F. The hot air blows through a hose and into a polypropylene "blanket" that lies atop the patient. Holes in the bottom of the blanket allow the air to jet onto the skin of the patient, warming convectively.

The technology generically referred to as forced-air warming ("FAW") is used in virtually every surgery in the United States lasting longer than 60 minutes. Bair Hugger holds a 90%+ share of the market.

Recent research

The most dramatic research directly linking Bair Hugger to peri-prosthetic joint infections ("PJIs") was conducted by McGovern *et al.* and published in the November 2011 issue of the *Journal of Bone and Joint Surgery Br.* This research shows that Bair Hugger FAW was linked to **a 3.8 times increase** in deep joint infections.

When the institution discontinued the use of FAW and switched to air-free conductive fabric warming, their peri-prosthetic joint **infection rate dropped 74%**, (3.1% ⇒ 0.8%) p=0.024, 1437 patients, 2.5 years.

The 74% decrease in PJIs resulting from discontinuing the use of FAW reported by investigators suggests that the majority of the 12,000-20,000 PJIs reported annually in the United States may be linked to FAW.

Five peer-reviewed, published studies have established that the 1000+ watts of rising heat produced by Bair Hugger blowers lift contamination from the floor of the operating room and deposit it in the sterile surgical field. The most recent study, by Legg *et al.* and published in the *Bone & Joint Journal,* showed a 217,000% increase in contamination over the wound site when Bair Hugger was used. The more contamination, of course, the more likely is an infection to occur.

The Opportunity

Medicare and SCIP ("Surgical Care Improvement Project") mandate active warming for most surgeries. It is highly likely, therefore, that any patient who received a new joint

within the past five years received forced-air warming. As noted, more than 90% of FAW is done by 3M's Bair Hugger.

PJIs are relatively common, occurring in 1-2% of hip and knee replacements: 12,000-20,000 PJIs per year in the US. Treatment requires ex-plantation of the device and several weeks of antibiotic therapy before re-implantation of a new hip or knee. PJIs are very expensive, often costing over $100,000 to treat, assuming no amputation. They are extraordinarily painful and permanently disabling. Cahill *et al.* showed that 12% of patients that survive a PJI with subsequent re-implantation rate their eventual quality of life as "worse than death."

If one estimates awards at $1 million and applies the six-year statute of limitations of Minnesota (3M's home), the **total liability exposure for 3M could exceed $100 billion.**

This is an opportunity for personal injury lawyers to force a major company to behave responsibly and to stop willfully exposing vulnerable patients to a higher risk of catastrophic injury. This may be one of the biggest product liability opportunities since the tobacco litigation of the '90s.

Tommy Walton Litigation:

On March 5, 2013, Kennedy Hodges filed an action in Harris County, Texas District Court against 3M Company, Arizant Healthcare, Inc. and Robert Prestera. The plaintiff is Tommy Walton, a 70-year-old Houston-area man.

The Original Petition is attached as Exhibit Z or may be downloaded at -- http://www.hcdistrictclerk.com/eDocs/Public/Search.aspx?ShowFF=1

Defendants have removed the case to federal court.

Kennedy Hodges' press release may be seen at KennedyHodges.com.

Mr. Walton underwent total hip replacement surgery at Houston Orthopedic Surgery Hospital in March 2011. An infection erupted. During the months that followed, Mr. Walton underwent 15 revisions. After treatment at Mayo Clinic in Rochester, Minnesota, the infection was controlled and an artificial hip was re-implanted. Mr. Walton remains seriously disabled, with one leg significantly shorter than the other.

In general, Mr. Walton alleges that Defendants' Bair Hugger patient warming system released heat at or below the surgical table. The convection currents created by such heat mobilized the bacteria that had settled to the floor of the OR, lifting it into the sterile field and depositing it into Mr. Walton's surgical wound. The bacteria caused the infection that injured Mr. Walton.

In addition, Mr. Walton alleges that the internal airflow paths of the Bair Hugger blowers are contaminated with pathogens that are blown into the sterile field.

3M/Arizant has been aware of these dangers for several years, Mr. Walton claims, and has no taken any action to protect surgical patients.

Arizant Healthcare is the division of 3M that manufactures Bair Hugger. 3M purchased Arizant from Court Square Capital in 2010 for $810 million and incorporated it into 3M's Infection Control Division.

The Petition, which demands damages in excess of $1,000,000 as well as exemplary damages, includes nine counts:

I. Negligence
II. Violation of the Texas Deceptive Trade Practices Act
III. Failure to Warn
IV. Defective Manufacturing and Design Defects
V. Breach of Express Warranty
VI. Breach of Implied Warranty
VII. Negligent Misrepresentation
VIII. Fraudulent Misrepresentation
IX. Fraudulent Concealment

Defendants answered the Petition. In addition to making general denials, Defendants asserted numerous affirmative defenses, including:

- Learned Intermediary Defense—In essence, Defendants asserted that they had adequately warned Mr. Walton's physicians regarding risk of injury.
- Informed Consent—In essence, Defendants asserted that Mr. Walton had consented to being exposed to the risk of injury after being adequately informed.

What is a peri-prosthetic joint infection ("PJI")? How does it compare to a surgical site infection ("SSI")?

The term "SSI" commonly refers to any infection that results from surgery. As with many multi-factorial disease processes, lumping the varieties together can cause confusion. A discussion of SSI makes much more sense if the term "SSI" is reserved for soft-tissue infections, which are differentiated from the PJI deep joint infections that can occur after total joint replacement surgery.

PJIs are catastrophic infections that occur after 1-2% of primary hip and knee replacement surgeries. According to the American Academy of Orthopedic Surgeons ("AAOS"), the incidence of PJIs is greater than 2% in the Medicare population and even higher for revision arthroplasty or "re-do" hip and knee surgeries.[1] It is estimated that there are 12,000-20,000 PJIs per year in the US. Considering the severity of this complication of total joint replacement surgery, 1-2% is an extraordinarily high incidence rate.

Information about PJIs

The implantation of foreign material in the joint (and probably any other location as well) fundamentally changes the pathophysiology of the infectious process. It has been shown that a *single* bacterium can cause a PJI, and it usually enters the wound as *airborne* contamination.[2-4]

PJIs manifest in one week to one year after the surgery. How can a single bacterium survive in the human body for a year with no symptoms and then emerge as a full-blown infection? The answer is *biofilm*.[5] In the presence of an implanted foreign material, the bacterium produces a coating of extracellular polymeric substances, also known as exopolysaccharide--biofilm that effectively protects it from both antibodies and antibiotics. The bacterium can go dormant for up to a year and then, when the conditions are favorable, multiplies into a deep infection.[6-8]

Because the biofilm-covered bacteria are coating the infected implant, the infection cannot be eradicated without removing the infected implant. The treatment of a PJI requires ex-plantation of the infected joint, IV antibiotics, prolonged hospitalization and, if the patient survives without amputation, re-implantation of the prosthetic joint at a later date. It costs more than $100,000 treat a PJI and can be much more than that if the infection results in amputation.

Even without amputation, these patients are often severely and permanently debilitated. Research by Cahill *et al.*, shows that patients who survive deep joint infections after joint replacement surgery without requiring amputation, never return to normal function or quality of life[9]:

> "Functional and health-related quality-of-life outcomes after infection are devastating for the patient. Infection had a great impact on physical functioning and ability to live independently and perform activities of daily living."

> "The overall quality of life in patients complicated with infection was poor…12% of the patients in the complicated group had their health state rated equivalent to or worse than death."

Ten to twelve thousand patients per year spending the rest of their lives with pain, suffering and disability, all because of preventable deep joint infections, constitutes a public health crisis.

Soft-tissue SSIs

A soft tissue SSI is fundamentally different from a PJI. An inoculum of more than one million bacteria (as opposed to one for PJIs) is required to cause an SSI, and the bacteria usually enter the wound from the adjacent skin or cut bowel.[10] SSIs manifest in one day to one month and are unable to "hibernate" and erupt later because the bacteria cannot produce effective biofilm in soft tissue. Without a biofilm coating, bacteria in soft tissue are exposed to both antibodies and antibiotics. Therefore, the size of the inoculum necessary to cause an infection in soft tissue is vastly larger. Because it takes more than one million bacteria to cause a soft tissue SSI, the airborne contamination in the operating room is virtually irrelevant for soft tissue SSIs. There simply is not that level of biological contaminants in the air. Finally, soft tissue SSIs are generally minor and easily treatable complications.

Arizant/3M, the manufacturer of Bair Hugger FAW, frequently cites to a colon surgery study by Kurz, claiming that it proves that FAW could not possibly cause an increased risk of deep joint infections. Kurz *et al.* reported a 66% reduction in soft tissue wound infections during colon surgery when the patients were warmed to normothermia with FAW (compared to 2°C hypothermic, non-warmed control patients).[11] Melling reported similar reductions in SSIs following breast and hernia surgery when patients were kept warm with FAW.[12]

The apparent paradox of FAW reducing SSIs on the one hand and increasing PJIs on the other is easy to explain. Colon, breast and hernia surgeries are all soft-tissue surgeries that are relatively unaffected by air contamination. The incidence of soft tissue SSIs are reduced due to the increased local blood flow, increased immune function and increased tissue oxygen levels resulting from the patient having a normal body temperature (normothermia), irrespective of how the normal temperature is achieved. In contrast, patient normothermia does not prevent PJIs because the open joint is cold even if the patient is warm, and the waste heat from FAW has been shown to contaminate the joint prosthesis leading to increased PJI risks.

References:

1. The diagnosis of periprosthetic joint infections of the hip and knee: Guideline and evidence report. American Academy of Orthopedic Surgeons, June 2010.
2. Lidwell OM et al. Bacteria isolated from deep joint sepsis after operation for total hip or knee replacement and the sources of the infections with *Staphylococcus aureus.* J Hosp Infect 1983;4:19-29.
3. Whyte W, Hodgson R, Tinkler J. the importance of airborne bacterial contamination of wounds. J Hosp Infect 1982;3:123-135.

4. Petty W, Spanier S, Shuster JJ. The influence of skeletal implants on incidence of infection. J Bone and Joint Surg 1985;67A:1236-1244.
5. Galanakos SP, et al. Biofilm and orthopaedic practice: the world of microbes in a world of implants. Orthopaedics and Trauma 2009;23(3):175-179.
6. Davis SC, et al. Microscopic and physiologic evidence for biofilm-associated wound colonization in vivo. Wound Repair and Regeneration 2008;16(1):23–9.
7. Lewis K. Riddle of Biofilm Resistance. Antimicrobial Agents and Chemotherapy 2001;45(4): 999–1007.
8. Parsek MR, et al. Bacterial biofilms: an emerging link to disease pathogenesis. Annual Review of Microbiology 2003;57:677–701.
9. Cahill JL et al. Quality of life after infection in total joint replacement. J Orthopaedic Surg 2008;16(1):58-65.
10. Elek SD, Cohen PE. The virulence of *Staphylococcus pyogenes* for man. A study of the problem of wound infection. Br J Exp Path. 1957;38:573-586.
11. Kurz A et al: Mild perioperative hypothermia increases the incidence of surgical wound infections and the duration of hospitalization in patients undergoing colon resection. *NEJM* 1996;334:1209-1215.
12. Melling AC, Leaper DJ. Effects of preoperative warming on the incidence of wound infection after clean surgery: a randomised controlled trial. Lancet 2001;358(9285):876-80.

How does Bair Hugger cause infections?

Summary: The waste heat from Bair Hugger warming mobilizes contaminants and germs from near the floor into convection currents of rising warm air. The contaminants are transported from the floor of the operating room, up and into the sterile surgical operating field above the table, contaminating the sterile field.

Background about OR ventilation:

It is an axiom in the operating room that the ventilation airflow should always be from the ceiling to the floor and never the reverse. Air should *never* rise in the vicinity of the surgical table, because the air below the level of the surgical table is well-known to be contaminated with dead skin cells and skin bacteria, shed from both the OR personnel and the patients. All modern operating room ventilation systems are designed to blow clean air from the ceiling vents downward onto the surgical site. Then the air is vented out of the room through wall vents located near the floor.

The flow direction is ceiling-to-floor only, clean-to-dirty, as mandated by ASHRAE 170– the relevant US standard for operating room ventilation, including laminar flow and ultra-clean operating rooms.[1]:

> 1.) Section 7.1.1.a: " Design of the ventilation system shall provide air movement that is generally from clean to less clean areas."
>
> 2.) Section 7.4.1.a: "The airflow shall be unidirectional, downwards…."

In other words, only a ceiling-to-floor airflow direction is allowed, from the clean HEPA filters in the ceiling, downward to the dirty floor and then out the vents near the floor.

The German DIN 1946-4:2008-12 standard ("DIN") for health care facility ventilation including laminar flow and ultra-clean operating rooms is regarded by most experts to be at least as rigorous and more detailed than the US ASHRAE 170 standard.[2]

Similar to ASHRAE 170, the DIN standard also has several sections prohibiting any upward airflow in the vicinity of the surgical table, in laminar flow and ultra-clean operating rooms:

> Section 5.2.1: "…the strict application of the dynamic barrier concept, which consists of flooding the area to be protected in the operating room with a low-turbulence flow…."
>
> Section 5.2.2: "…low-turbulence flow (LTF) throughout the protected area, in particular a vertical inflow into the protected area via [the ceiling mounted] HEPA filter [plenum]…."

> Section B.2.1.2: "The test aerosol shall exhibit a uniform outflow. Neither localized disturbances of the emitted aerosol nor inhomogeneities of the outflow behavior shall be detectable at any position below the LTF outlet."
>
> Section B.2.2.2: "Neither a reversal of the flow direction nor a lifting effect shall be detectable at any position."

The DIN standard does not specifically name FAW as being prohibited for use in laminar flow and ultra-clean operating rooms. However, Section 6.8 says that

> "…decentralized heating and cooling devices with a *convective effect* are not permitted [in laminar flow and ultra-clean operating rooms]." (emphasis added)

In other words, only ceiling-to-floor airflow is allowed.

Since the introduction of FAW in 1988, it has been assumed that FAW's waste air (40 cfm) simply mixed with the thousands of cfm of operating room ventilation air and was harmlessly exhausted out of the room. If it were just waste air, this assumption would be accurate. However, in late 2008 researchers at Augustine Biomedical + Design ("ABD"), realized the importance of the 800-1000 watts of waste heat that was exhausting with the waste air. It quickly became apparent that the waste heated air trapped under the surgical drape is pushed by the mass effect of 40 cfm of waste air until it underflows the lower edge of the surgical drape hanging down along the sides of the table.[3]

The researchers showed that when the heated waste air finally escapes near the floor, it mixes with the contaminated air that is normally resident near the floor. The waste FAW heat and air, mixed with the floor air, then rises along the sides of the surgical table directly into the downward ventilation airflow. For years everyone overlooked the simple fact that heat rises. It does not matter if the ventilation airflow is laminar and/or ultra-clean (for implant surgeries) or conventional, the waste heat can easily rise into the downward airflow mobilizing the contaminated floor air with it. The contaminated floor air ends up on top of the surgical table, directly in the sterile field. Especially in the presence of ventilation airflow obstructions such as the surgical light, the surgeon or the Mayo stand, the rising convection currents of waste heat are unstoppable. The convection currents of rising waste FAW heat were first publicly reported in October 2009, at the Annual Meeting of the American Society of Anesthesiologists (ASA) and posted on the www.Heat-rises.blogspot.com website shortly thereafter.

Three mechanisms of sterile surgical field contamination by the waste FAW heat have been documented in video research—all of which have been corroborated by the peer-reviewed published studies noted below:

> 1. Waste heat vents from under surgical table and heats the contaminated air normally resident near the floor. The heated air forms convection currents that rise along side the table into the downward ventilation airflow and end up on top of the table in the sterile surgical field.

(McGovern, Legg)

2. Waste heat vents from head end of the table, rising and traversing over the top of the anesthesia drape (ether screen) and into the surgical field. (link)
(McGovern, Belani)

3. Waste heat radiates through the surgical drape inducing vortexes on the surgical side of the anesthesia drape. The vortexes act exactly like mini-tornados, sucking contaminates off the floor and transporting them into the sterile surgical field.
(Legg, Dasari)

These phenomena have been confirmed by the following published studies:

• McGovern et al. Forced-air warming and ultra-clean ventilation do not mix. *Journal of Bone and Joint Surgery-Br.* 2011;93(11):1537-1544.

Orthopedic surgeons investigated the capacity of patient warming devices to disrupt the ultra-clean airflow system. They compared the effects of Bair Hugger FAW and HotDog conductive fabric warming on operating theatre ventilation during simulated hip replacement and lumbar spinal procedures using a mannequin as a patient.

"Excess heat from FAW resulted in the development of hot-air convection currents between the surgeon's body and the operating table, that transported [contaminated] floor-level air upwards and into the surgical site."

"…forced-air warming mobilized under-drape air so that it passed over the anaesthesia/surgery drape and into the surgical site, but conductive fabric warming did not have a mobilizing effect. "

"Forced-air warming was found to have a significant and disruptive impact on the clean airflow patterns over the surgical site compared to conductive fabric warming, which had no noticeable effect. Further, forced-air warming established convection currents that mobilized resident air from non-sterile areas such as the floor and under the anaesthesia/surgery drape into the surgical site."

• Legg et al. Do forced air patient-warming devices disrupt unidirectional downward airflow? *Journal of Bone and Joint Surgery-Br,* 2012;94-B:244-256.

During simulated lower-limb arthroplasty, the investigators studied the temperature and the number of particles detected over the surgical site comparing Bair Hugger FAW with HotDog conductive warming.

"Forced air warming resulted in a significant mean increase in the temperature (1.1°C *vs* 0.4°C, $p<0.001$) and the number of airborne particles (1038.2 *vs* 274.8, $p=0.0087$) over the surgical site when compared to the radiant warming [or control], which raises concern as bacteria are known to require particles for transport."

"The results do suggest that the downward flow of air is disrupted, as the warming device was lower than the surgical site."

• Dasari et al. Effect of forced air warming on the performance of operating theatre laminar flow ventilation. *Anaesthesia*, 2012; 67:244-249.

The researchers investigated whether the floor-to-ceiling temperatures over a draped manikin in a laminar-flow theatre differed when using three types of warming devices: Bair Hugger FAW, HotDog conductive fabric warming and an underbody resistive mattress.

The foot-end of the surgical drape was intentionally raised to create an air channel that directed the forced-air exhaust out of the ventilation field—allowing the heat radiating through the drapes to be studied.

> "With forced-air warming, mean (SD) temperatures were significantly elevated over the surgical site *vs* those measured with the conductive blanket (+2.73 (0.7)°C; p<0.001)…"
>
> "We conclude that forced-air warming generates convection current activity in the vicinity of the surgical site."

• Belani et al. Patient warming excess heat: The effects on orthopedic operating room ventilation performance. *Anesthesia & Analgesia,* 2012 Jul 19, Epub ahead of print.

The investigators studied the effects of two popular patient warming technologies: FAW vs conductive fabric vs control on ventilation performance in an orthopedic operating room with a mannequin draped for total knee replacement. Ventilation performance was assessed by releasing neutrally buoyant detergent bubbles into the nonsterile region under the head-side of the anesthesia drape. They then tracked whether the excess heat could mobilize the bubbles over the drape and into the sterile surgical field.

> "…forced air warming was found to establish convection currents that mobilized resident air from the nonsterile areas (under the anesthesia drape) upward and into the surgical site. The clinical concern is that convection currents may mobilize underdrape contaminants into the surgical site and/or impede the ventilation systems' ability to clear contaminants from the surgical site."
>
> "Excess heat from forced air warming resulted in the disruption of ventilation airflows over the surgical site, whereas conductive patient warming devices had no noticeable effect on ventilation airflows."

• Legg et al. Forced-air patient warming blankets disrupt unidirectional airflow. *Bone and Joint Journal,* 2013;95-B:407-410.

The investigators attempted to visualize airflow in the theater over a simulated total knee replacement using neutral-buoyancy bubbles. Smoke particles were then released below the operating table and detected above the surgical site. The heat radiating through the surgical drape created vortexes that drew particles from near the floor, up and into the sterile surgical field.

> "...waste heat from the poorly insulated forced-air warming blanket increased the air

> temperature on the surgical side of the drape by > 5°C. This created convection currents that rose against the downward unidirectional airflow, causing turbulence over the patient."
>
> The convection currents increased the particle concentration 2000-fold (2,174,000 particles/m^3 for forced-air warming vs 1000 particles/m^3 for air-free HotDog warming) by drawing potentially contaminated particles from below the operating table into the surgical site.
>
> "...forced-air warming...can significantly disrupt unidirectional air flow and draw particles from the potentially contaminated area below the sterile field. This is a concern."

In addition the these published studies, the April 2013 issue of *Health Devices* contains guidance from ECRI Institute regarding the use of forced-air warming in surgery.[4] ECRI is an independent scientific organization that advises its members regarding medical devices, drugs and procedures.

After reviewing the published, peer-review articles cited above that address the consequences of the convection currents created by the waste heat produced by forced-air devices, ECRI stated:

> "The disruption of air-flow patterns is particularly worrisome in laminar-flow and ultraclean ORs, in which a wide variety of implant surgeries are performed."
>
> "This is especially concerning during orthopedic surgeries because contamination of the surgical site may present a greater risk of developing a PJI [periprosthetic joint infection], which is harder to treat and resolve than would be the case with SSIs [surgical site infections] in general."

These new studies and ECRI's expressions of concern make clear that the unrecognized causative factor of PJIs is forced-air warming—specifically, Bair Hugger forced-air warming.

The apparent increase in deep joint infection rates in laminar flow ventilation operating rooms over the past 20 years may be a result of the introduction of FAW to orthopedic surgery. In the 1980s, Lidwell reported the results of the only large randomized controlled trial (RCT) that has studied the benefits of laminar flow ventilation for total joint replacement surgery.[5] He showed that laminar flow ventilation plus antibiotics resulted in reducing the deep joint infection rate to only 0.4% . Over the next 20 years, OR ventilation systems improved and protocols for orthopedic surgery became much more rigorous. For example, protocols now routinely require the use of "space suits" by the surgeons and severely limit personnel movement in the OR. Despite the improved surgical protocols and equipment, two studies from the 2000s showed that the deep joint infection rates in laminar flow operating rooms had increased by a factor of 3.5 times, to 1.4%, a full percentage point greater than the 1980s.[6,7]

While deep joint infections typically occur only in about 1-2% of total joint replacement surgeries, the incidence can be much higher at any given hospital. Ironically, many of the

hospitals reporting higher than "normal" deep joint infection rates are well known for their high quality care. This seems to indicate that reducing the deep joint infection rate is not as simple as strictly following accepted operating room protocols -- these high quality hospitals are already doing that. Logically, there must be a previously unrecognized causative factor that is an accepted part of the current protocol. By interesting coincidence, during the past 20 years FAW has progressively gained acceptance, and by the 2000s was generally recognized as the "standard of care" for all surgical procedures. The recent evidence that FAW disrupts the operating room ventilation with contaminated air from the floor strongly suggests that FAW is the "accepted part of the current protocol" that is responsible for the very significant increase in deep joint infection rates documented over these years.

In summary, there are now five peer reviewed published studies proving that the waste heat from Bair Hugger warming causes convection currents of rising contaminated air by at least three different mechanisms. It is irrefutable that the waste heat from Bair Hugger warming causes significant increases in the number of contaminates (germs and particles) in the sterile surgical field.

References:

1. Guidelines for Design and Construction of Health Care Facilities, Facility Guidelines Institute. 2010
2. Sessler DI, Olmsted RN, Kuelpmann R. Forced-air warming does not worsen air quality in laminar flow operating rooms. *Anesth Analg* 2011;113:1416-21.
3. Heat-rises.blogspot.com
4. Forced-air warming and surgical site infections, ECRI Institute. health Devices 2013;42-4:122-125.
5. Lidwell OM. Air, antibiotics and sepsis in replacement joints. J. Hosp Infect. 1988; 11 Suppl C:18-40.
6. Brandt C, Hott U, Sohr D, Daschner F, Gastmeier P, Ruden H. Operating room ventilation with laminar airflow shows no protective effect on the surgical site infection rate in orthopedic and abdominal surgery. Ann Surg. 2008;248(5):695-700.
7. Knobben BAS, et al. Evaluation of measures to decrease intra-operative bacterial contamination in orthopedic implant surgery. J Hosp Infection 2006;62:174-180.

Bair Hugger's internal contamination: An additional risk

Summary: Research shows that nearly all Bair Hugger FAW blowers are internally contaminated with bacteria and mold colonies, and are aerosolizing large numbers of bacteria and mold out of the hose and into the operating room.

Three recently published peer reviewed studies show that nearly all Bair Hugger blowers are contaminated with bacteria and mold on their inner airflow paths.[1-3] The research also shows that the majority of these blowers are blowing large quantities of germ-sized particles, presumably germs, out of their hoses and into the operating rooms.[1-3]

The reason: Arizant intentionally reduced the efficiency of its air filters.
On Sept 6, 2000, Arizant filed a 510k identified as K001149 for their Model 750 blower. In that 510(k) they represented to the FDA that the inlet filter of the Model 750 was "HEPA," an improvement over the ".2 micron filter" of the predicate Model 505. The US CDC defines a HEPA (High Efficiency Particulate Air) filter as an air filter that removes more than 99.97% of particles 0.3 microns or larger.

At some point, Arizant management apparently concluded that they needed to increase the airflow from the 100,000-plus blowers currently in service (that they own). Rather than replacing the blowers with higher airflow models, they instead apparently decided to reduce the inlet filter efficiency in order to increase the airflow.

Research by Reed published showed that the Model 750 filters that were purported to be HEPA quality (99.97% efficient) were in fact only 63.8% efficient.[2] Research by Albrecht published in reported that the 0.2 micron filters on the Model 505 blowers had been reduced from 93.8% efficiency to 61.3%.[1]

These decreases in filter efficiency from HEPA and 93.8%, to 63.8% and 61.3%, could not have been legally accomplished by a "letter to file." Substantial decreases in safety require a submission of a new 510(k) for formal review, and this was never done. Therefore, Bair Hugger warming in its current configuration is "adulterated." The results of Arizant decreasing the filter efficiency of their blowers has also been recently studied:

• Albrecht M, Leaper D et al. Forced-air warming blowers: An evaluation of filtration adequacy and airborne contamination emissions in the operating room. *Am J Infect Control* 2011;39:321-8.

The internal air path surface was swabbed and cultured, and hose outlet particle counts were performed on 52 forced-air warming devices (all with the model 200708C filter) to assess internal microbial buildup and airborne contamination emissions. The retention efficiency of the inlet filters was assessed using a monodisperse sodium chloride aerosol.

Micro-organisms were cultured from the internal airflow paths of 92.3% of the 52 blowers tested, including *Staphylococcus aureus* (13.5%) and methcillin resistant *Staphylococcus aureus* (MRSA) (1.9%).

58% of the Bair Hugger blowers tested were found to be internally generating and emitting significant levels of airborne contaminants >0.3µm in size (germ size), up to 35,272 particles per ft^3 of air (80 million particles per hour).

The 0.2 micron filters on the Model 505 blowers had a filtration efficiency of 61.3%.

• Reed M et al. Forced Air Warming Design: An Evaluation of Intake Filtration, Internal Microbial Build-Up, and Airborne-Contamination Emissions. *AANA Journal* Accepted 2012: in press.

Reed *et al.* sampled twenty-three forced air warming devices (Bair Hugger Model 750, Arizant Healthcare) in daily hospital use for internal microbial build-up and airborne-contamination emissions via swabbing and particle counting. They also rated the intake filtration efficiency of the Bair Hugger Model 750 forced air warming devices using a monodisperse sodium chloride aerosol in a laboratory setting.

Micro-organisms were cultured from the internal airflow paths of 100% of the 23 blowers tested, including *Staphylococcus aureus* (74%), mold (26%) and *Micrococci* (9%).

100% of the blowers tested were found to be emitting internally generated airborne contaminants >0.3µm in size (germ size), up to 112,000 particles per ft^3 of air (300 million particles per hour).

In the most contaminated blower, the emitted particle count was 40 times greater than the intake particle count, and the intake particles had to go through a 64% efficient inlet filter. Therefore, nearly all of these particles (germs) were generated inside the blower.

The Model 750 inlet filters that were purported to be HEPA quality (99.97% efficient) were in fact only 63.8% efficient.

• Albrecht M and Leaper D et al. "Forced-air warming: a source of airborne contamination in the operating room?" *Orthopedic Reviews* 2009;1:e28.

These researchers measured the emission of viable and non-viable forms of airborne contamination from 25 FAW blowers in the operating room with a laser particle counter. Filtration efficiency was calculated as the reduction in particulate concentration in the distal hose airstream relative to that of the intake. Microbial colonization of the FAW blower's internal hose surfaces was assessed by culturing the microorganisms.

24% of FAW blowers were emitting significant levels of internally generated airborne contamination in the 0.5 to 5.0 µm size range.

> The particle size-range-specific reduction in efficiency could not be explained by the filtration properties of the intake filter. Instead, the reduction was found to be caused by size-range-specific particle generation within the FAW blowers.
>
> Microorganisms were detected on the internal air path surfaces of 94% of FAW blowers.

While these three studies are the most compelling, as early as 1997 studies have reported that Bair Hugger blowers were internally contaminated with bacteria that can cause surgical site infections (SSI):

• Avidan MS et al., "Convection warmers--not just hot air," *Anaesthesia* 52, no. 11 (November 1997): 1073-6.

> Cultured approximately 5 convective warmers by 1) swabbing the inside of the hose and 2) blowing the distal hose end air stream over agar plates. Heavy colonization was detected in the hose swabs and nearly all of the agar plates grew organisms captured from the air.

• Bernards AT et al., "Persistent Acinetobacter baumannii? Look inside your medical equipment," *Infection Control and Hospital Epidemiology : the official journal of the Society of Hospital Epidemiologists of America* 25, no. 11 (November 2004): 1002-4.

> Traced an outbreak of drug resistant *acinetobacter baumannii* to dust sampled from the inside of Bair Hugger and respiratory ventilators. Cleaning the inside of the equipment stopped future outbreaks.

• Baker N, King D, and Smith EG, "Infection control hazards of intraoperative forced air warming," *The Journal of Hospital Infection* 51, no. 2 (June 2002): 153-4.

> Cultured a convective warming unit routinely used in ultra clean orthopedic theaters by swabbing the exterior and interior of the device. Heavy colonization was detected in all samples. Based upon prior research and their own data, Baker et al. recommended against the use of convective warming for orthopedic procedures based upon the elevated risk of SSI.

The management at Arizant/3M is most certainly aware of these studies showing internal contamination of their blowers and that the blowers are contaminating the operating rooms with these germs. It may be alleged in litigation that the company has acted irresponsibly in light of this patient safety hazard.

<u>What could 3M have done to make their contaminated blowers safer?</u>

1. FAW air-circulation systems should be required to meet all aspects of the American Society of Heating, Refrigeration and Air-conditioning Engineers ("ASHRAE") Standard 170. Since "internal cleaning" is impossible with the current

blower designs, blowers should be equipped with HEPA filters on both the inlet and outlet sides. Otherwise pathogens will continue to breed inside the blowers and, without a hose-end filter, will be blown into the sterile field.

2. As recommended by the researcher experts,[1-3] adding a HEPA filter to the hose-end will also bring the FAW blowers into conformity with operating room ventilation standards promulgated by the Hospital Infection Control Practices Advisory Committee of the National Center for Infectious Diseases ("HICPA").

3. Because the internal airflow paths of FAW blowers cannot be accessed for cleaning on site, contaminated blowers should be recalled from the field and decontaminated before being put back into service.

4. Warning labels should be required, identifying the safety risk to patients and the hospital staff of internal blower contamination.

What did 3M do in response to this research? Nothing, except to deny that the problem even existed. Specifically, they have not warned their customers, they have not notified the FDA and they have not recalled the blowers for cleaning. Perhaps most importantly, they have not added hose-end filters to assure that only germ-free air is emitted from their blowers.

References:

1. Albrecht M, Leaper D et al. Forced-air warming blowers: An evaluation of filtration adequacy and airborne contamination emissions in the operating room. *Am J Infect Control* 2011;39:321-8.
2. Reed M et al. Forced Air Warming Design: An Evaluation of Intake Filtration, Internal Microbial Build-Up, and Airborne-Contamination Emissions. *AANA Journal* 2012: in press.
3. Leaper D et al. Forced-air warming: a source of airborne contamination in the operating room? *Orthopedic Rev.* 2009;1(2):e28.

"The Chain of Infection": The science of linking contamination of the sterile surgical field by the waste heat from Bair Hugger to peri-prosthetic joint infections.

The Chain of Infection is a well-known model that is used to understand the etiology of infections.[1] The Chain of Infection model is frequently used by the US Centers for Disease Control (CDC) or state departments of public health to back-track the cause of a given infection. Each link in the chain must be present for an infection to occur. Each of these links will be discussed as they apply to PJIs.

The six links in the Chain of Infection
1. Infectious Agent
2. Reservoir
3. Portal of Exit
4. Mode of Transmission
5. Portal of Entry
6. Susceptible Host

For PJI, five of these six links in the chain have been well documented for decades. The missing link has been the Mode of Transmission—how do the infectious agents get from the floor up into the open wound? Recently published research has closed the missing link by showing that the waste heat from forced-air warming mobilizes operating room contaminants into the surgical wound.

1. Infectious Agent: A microbial organism with the ability to cause disease.

The CDC has said that PJIs are primarily caused by *Staphylococcus aureus*, coagulase-negative staphylococci and gram-negative bacilli, predominantly skin bacteria.[2] Obviously, for any individual patient, the start of the chain is the specific bacterium cultured from the infected joint.

2. Reservoir: A place within which microorganisms can thrive and reproduce.

It has been shown that most of the bacteria in the operating room are shed from the skin of the surgical staff.[3-8] Presumably, some are also shed from the patient. Dispersed airborne skin bacteria can also originate in the perineal region of the staff, including vaginal and rectal carriage.[2,9-16]

Recently published research has shown that nearly all forced-air blowers are contaminated with bacteria growing on their internal airflow paths.[17-24] These bacteria have been shown to be predominantly of skin origin like most of the airborne contaminants in the operating room.[17,18] The blowers suck in the contaminated air and the low efficiency inlet filters fail to block contaminating bacteria from entering the blower where they can stick to the plastic parts and grow.[17-18]

3. Portal of Exit: A place of exit providing a way for a microorganism to leave the reservoir.

In this chain, there are multiple portals of exit from the reservoirs:

a. Simple shedding.
The average person sheds one billion skin cells per day and up to 10% of these have one or more bacteria attached.[23-26] The skin cells have been called "skin rafts" because they ride the air currents in the operating room, transporting the attached bacteria much like a flying carpet.[5] Free-floating bacteria can also be shed from the staff.

b. "Space suit" exhaust.
The internal ventilation systems of space suits worn by surgeons usually vent from the open bottom of the gown. The shed skin cells and bacteria follow the internal air currents down and out the bottom of the gown near the floor.[6]

c. Rectal and vaginal shedding.
It has been shown that a relatively large percentage of airborne organisms in the operating room are shed from the perineal region of the staff.[2,9-16] Plastic underwear have been shown to decrease the release of this shed material.[9,27] Movement of the staff within their scrub pants has been shown to both increase exfoliation of dead skin and create a bellows or pumping action that blows desquamated skin cells and bacteria out of the legs of the garment and into the air near the floor.[28-31]

d. Aerosolized bacteria out the hose of the forced-air blower.
One study showed that 58% of the FAW blowers tested were emitting large quantities of internally generated (grown) germ-sized particles from the hose end.[17] In another study, 96% of the blowers were emitting up to 300 million germ-sized particles per hour.[18]

4. Mode of Transmission: Method of transfer by which the organism moves or is carried from one place to another.

It has been shown in many studies that bacteria in the operating room can ride air currents.[29,30,32,33] Because the general airflow of the ventilation air is from ceiling to floor, it is not surprising that the shed skin particles and bacteria concentrate near the floor.[30,32,34,35] Additionally, contaminants that have settled on the floor can be re-aerosolized by the movement of the personnel in the room.[30,34,36,37]

The missing link in the Deep Joint Chain of Infection has been an adequate explanation of how airborne contaminants that naturally accumulate near the floor can rise up into the sterile surgical field.

It has been shown that small convection currents of heat rising from the head of the surgeons or from the surgical lights can penetrate the downward ventilation airflow.[30,38] However, heat rising at or above head level is basically clean air and, therefore, is immaterial to the issue of surgical contamination.

The waste heat from forced-air warming is a totally different situation.[39-43] First, there are nearly 1000 watts of waste heat from FAW that create massive convection currents (compare that with about 100 watts radiating from the whole body of the surgeon).[44]

Second, the waste heat from a lower body or underbody FAW blanket escapes from under the surgical drapes at their lower edge near the floor.[44] In this location, the waste heat is mixing with the floor air that has the highest concentration of airborne contaminants. The waste FAW heat warms the contaminated floor air. Then the heated floor air rises alongside the surgical table, directly into the downward ventilation airflow and into the sterile surgical field above the wound.[39-41,43]

Alternatively, the waste heat from an upper body blanket has been shown to rise along the anesthesia screen. If a surgical light is positioned in the typical location over the chest of the patient, the rising warm waste air crosses over the anesthesia screen into the "dead zone" under the light and then into the sterile field.[44] Contaminants from the anesthesia provider as well as the patients head and arms have been shown to ride the convection current of waste heated air into the sterile surgical field.[39-41,43]

> "...forced-air warming established convection currents that mobilized resident air from non-sterile areas such as the floor and under the anaesthesia/surgery drape into the surgical site." McGovern et al[39]

The waste heat from a torso type blanket has been shown to radiate through the sterile surgical drape, heating the air in the surgical field next to the anesthesia drape (ether screen). The heated air in the "dead zone" that forms next to the ether screen starts to rotate and soon becomes a vortex that resembles a tornado when visible bubbles are added to it. Legg *et al.* have demonstrated that these vortexes can actually suck contaminant particles up from the floor and deposit them into the sterile surgical field. 2000 times more particles were detected in the surgical field with FAW than with air-free conductive warming.[43]

With five peer-reviewed, published studies documenting this effect, it is now irrefutable that the massive amount of waste heat from FAW venting near the floor grossly disrupts the protection of the surgical ventilation system.[39-43] The waste heat has been clearly shown to mobilize contaminants from the floor and head end of the patient into the sterile surgical field.

This is the missing link in the Deep Joint Chain of Infection.

5. Portal of Entry: An opening allowing the microorganism to enter the host.

The surgical wound, of course, is the portal of entry. It has been shown that the concentration of airborne contaminants in the surgical field is directly proportional to the concentration of contaminants in the wound.[37,46-54] The waste heat from FAW clearly increases the airborne contamination in the sterile surgical field.[39-43,55,56] Contaminating the air in the surgical field with contaminated air from the floor inevitably leads to increased contamination of the wound.

6. Susceptible Host: A person who cannot resist a microorganism invading the body, multiplying and resulting in infection.

The total joint arthroplasty patient is susceptible to even small numbers of airborne contaminants because of the implanted foreign materials. As was previously noted, in the presence of a foreign body, a single bacterium can cause a PJI and it is usually from airborne contamination.[57-59] The Periprosthetic Joint Chain of Infection is complete.

References:

1. Principles of Epidemiology in Public Health Practice, 3rd Edition, Centers for Disease Control and Prevention, Atlanta, GA
2. Mangram A, et al. CDC -- Guideline for Prevention of surgical site infection, 1999. AJIC 1999;27:97-132.
3. Lidwell, OM et al. Infection and sepsis after operations for total hip or knee replacement: influence of ultraclean air, prophylactic antibiotics and other factors. J. Hyg (Camb.) 1984;93:505.
4. Ayliffe GA et al. Role of the environment of the operating suite in surgical wound infection. Rev Infect Dis 1991;13(Suppl 10):S800-4.
5. Doig CM. The effect of clothing on the dissemination of bacteria in operating theaters. Br J Surg 1972;59:878-881.
6. Whyte W et al. Bacterial dispersion in relation to operating room clothing. J Hyg (Camb) 1976;76:367-378.
7. Burke JF. Identification of sources of staphylococci contamination of the surgical wound during operation. Ann Surg 1963; 158:898-904.
8. Favero MS, et al. Comparative levels and types of microbial contamination detected in industrial clean rooms. Appl Microb 1966;14:539-551.
9. Bethune D. W., et al. Dispersal of *Staphylococcus aureus* by patients and surgical staff. Lancet 1965;285:480–483.
10. Berkelman RL, et al. Streptococcal wound infection caused by a vaginal carrier.JAMA1982;247:2680-2.
11. Schaffner W, et al. Hospital outbreak of infections with group A streptococci traced to an asymptomatic anal carrier. N Eng J Med 1969;280:1224-5.
12. Stamm W, et al. Wound infection due to group A streptococcus traced to a vaginal carrier. J Infect Dis 1978;138:287-92.
13. McIntyre DM. An epidemic of Streptococcus pyogenes puerperal and postoperative sepsis with an unusual carrier site- the anus. Am J Obstet Gyn 1968; 101:308-14.
14. May KR, et al. Bacterial dispersion from the body surface. In: Airborne Transmission and Airborne Infection (Ed. By JF Hers and KC Winkler), p-426.l Oosthoek, Utrecht.
15. Hill J, et al. Effect of clothing on dispersal of Staphylococcus aureus by males and females. Lancet 1974;ii:1131.
16. Mitchell NJ, et al. Clothing design for operating room personnel. Lancet 1974;ii:1133.
17. Albrecht M, Leaper D et al. Forced-air warming blowers: An evaluation of filtration adequacy and airborne contamination emissions in the operating room. Am J Infect Control 2011;39:321-8.
18. Reed M et al. Forced Air Warming Design: An Evaluation of Intake Filtration, Internal Microbial Build-Up, and Airborne-Contamination Emissions. AANA Journal 2012: in press.
19. Leaper D et al. Forced-air warming: a source of airborne contamination in the operating room? Orthopedic Rev. 2009;1(2):e28.
20. Avidan MS, et al. Convection warmers--not just hot air. Anaesthesia. 1997;52(11):1073-107.
21. Baker N, et al. Infection control hazards of intraoperative forced air warming. Hosp. Infect. 2002;51(2):153-154
22. Bernards AT,et al. Persistent Acinetobacter baumannii? Look inside your medical equipment. Infect.Control Hosp. Epidemiol. 2004;25(11):1002-1004.
23. Jansen LH, et al. Improved fluorescence staining technique for estimating turnover of the human stratum corneum. Br J Derm 1974;90:9-12.
24. MacIntosh CA, et al. The dimensions of skin fragments dispersed into the air during activity. J Hyg (Camb) 1978;81:471-479.

25. Noble WC, et al. The size distribution of airborne particles carrying micro-organisms. J Hyg (Camb) 1963;66:385-391.
26. Noble WC, et al. Dispersal of skin microorganisms. Br J Derm 1975;93:447-485.
27 Blowers R, et al. Design of operating room dress for surgeons. Lancet 1965;ii:681-683.
28. Clark RP. The aerodynamics of warmth and cleanliness. Clothing Res. J. 1974;2:55–67.
29. Clark RP, et al. The generation of aerosols from the human body. In Airborne transmission and airborne infection (eds Hers J. F. P. H., Winkler K. C., editors.). 1973. Utrecht, The Netherlands: Oosthoek Publishing Co.
30. Clark RP, et al. Some aspects of the airborne transmission of infection. J Royal Soc Interface 2009;6 (Suppl 6):S767-782.
31. Whyte W et al. Reduction of microbial dispersion by clothing. J Parenteral Sci & Tech 1985;39:51-60.
32. Eames I, et al. Movement of airborne contaminants in a hospital isolation room. J Royal Soc Interface 2009;6 (Suppl 6):S757-766.
33. Davies RR, et al. Dispersal of bacteria on desquamated skin. Lancet. 1962;ii:1295.
34. Hambraeus A, et al. Bacterial contamination in a modern operating suite. 3. Importance of floor contamination as a source of airborne bacteria. J Hyg (Camb.) 1978;80:169-174.
35. Pasquarella C, et al. The index of microbial air contamination. J Hosp Infect 2000;46:241-256.
36. Bagnold R. A. The re-entrainment of settled dusts. Int. J. Air Pollut. 1960;2:357.
37. Friberg B, et al. Correlation between surface and air counts of particles carrying aerobic bacteria in operating rooms with turbulent ventilation: an experimental study. J Hosp Infect 1999;42(1):61-8.
38. Whyte W, et al. The effect of obstructions and thermals in laminar-flow systems. J Hyg (Camb) 1974;72:415-423.
39. McGovern et al. Forced-air warming and ultra-clean ventilation do not mix. J Bone and Joint Surg-Br. 2011;93(11):1537-1544.
40. Legg et al. Do forced air patient-warming devices disrupt unidirectional downward airflow? J Bone and Joint Surg-Br. 2012;94-B:254-6.
41. Belani et al. Patient warming excess heat: The effects on orthopedic operating room ventilation performance. Anesthesia & Analgesia 2012;X:X (prepublished online).
42. Dasari et al. Effect of forced air warming on the performance of operating theatre laminar flow ventilation. Anaesthesia 2012;67:244-249.
43. Legg et al. Forced-air patient warming blankets disrupt unidirectional airflow. *Bone and Joint Journal,* 2013;95-B:407-410.
44. Heat-rises.blogspot.com
45. Lidwell OM. Air, antibiotics and sepsis in replacement joints. J. Hosp Infect. 1988; 11 Suppl C:18-40.
46. Lidwell OM. Clean air at the operation and subsequent sepsis in the joint. Clin Orthop 1986; 211: 91-102.
47. Lidwell OM, et al. Airborne contamination of wounds in joint replacement operations: the relationship to sepsis rates. J Hosp Infect 1983;4:111.
48. Ritter MA. Operating room environment. Clin Orthop 1999; 369:103.
49. Whyte W. The role of clothing and drapes in the operating room. J Hosp Infect 1988;11(Suppl C):2-17.
50. Gosden PE, et al. Importance of air quality and related factors in the prevention of infection in orthopaedic implant surgery. J Hosp Infect 1998; 39(3):173-80.
51. Stocks GW, et al. Predicting bacterial populations based on airborne particulates: a study performed in nonlaminar flow operating rooms during joint arthroplasty surgery. Am J Infect Control 2010;38(3):199-204.
52. Howorth H. Prevention of airborne infection in operting room. J Med Sci and Tech 1987;11(5):263-266.
53. Lidwell OM, et al. Effects of ultraclean air in operating room on deep sepsis in the joint after total hip or knee replacement: A randomized study. Br Med J 1982;285:10-14.
54. Nelson JP. The operating room environment and its influence on deep wound infection in the hip. Proceedings of the 5th Scientific Meetings of Hip Society. CV Mosby, St. Louis.
55. Tumia N, et al. Convection warmners—a possible source of contamination in laminar airflow operating theatre? J Hosp Infection 2002;52:171-4.
56. Scherrer M. Hygiene and room climate in the operating room. Min Invas Ther & Allied Tech 2003;12(6);293-299.
57. Lidwell OM et al. Bacteria isolated from deep joint sepsis after operation for total hip or knee

replacement and the sources of the infections with *Staphylococcus aureus.* J Hosp Infect 1983;4:19-29.

58. Whyte W, Hodgson R, Tinkler J. the importance of airborne bacterial contamination of wounds. J Hosp Infect 1982;3:123-135.
59. Petty W, Spanier S, Shuster JJ. The influence of skeletal implants on incidence of infection. J Bone and Joint Surg 1985;67A:1236-1244.

The McGovern study: linking Bair Hugger to peri-prosthetic joint infections.

McGovern et al. Forced-air warming and ultra-clean ventilation do not mix. *J Bone and Joint Surg-Br.* 2011;93(11):1537-1544.

Drs. McGovern and Reed, orthopedic surgeons in the Northumbria Healthcare NHS Trust in the UK, **reported a dramatic 74% decrease in PJIs when they discontinued using Bair Hugger warming during total joint replacement surgery**. They switched to HotDog air-free warming, maintaining their new low infection rate. This research shows that Bair Hugger FAW was linked to a 3.8 times increase in deep joint infections.[1] When the institution discontinued the use of FAW and switched to air-free conductive fabric warming, their deep joint infection rate dropped 74%, (3.1% ⇒ 0.8%) p=0.024, 1437 patients, 2.5 years. These results are highly statistically significant.

> "Disruption [by FAW] in the ventilation of the surgical site was associated with significantly higher risks of joint sepsis…"
>
> "The risks of developing deep infections were significantly greater for patients… treated with forced-air [3.1%] *versus* conductive fabric [0.8%] warming."
>
> "A significant increase in deep joint infection, as demonstrated by an elevated infection odds ratio (3.8, p=0.024), was identified during a period when forced-air warming was used compared to a period when conductive warming was used. Air-free warming is, therefore, recommended over forced-air warming for orthopedic procedures."

The article also clearly demonstrates the mechanism by which FAW increases the infection risk—an unintended consequence of the rising waste heat. These researchers were totally financially independent - no payments of any kind were made to the investigators or their department.

The McGovern research was critiqued by ECRI in the April 2013 issue of *Health Devices*.[1] ECRI stated that the McGovern infection data was "not conclusive" for several reasons including:

- The study was retrospective rather than the randomized control trial (RCT) design that is preferred.
- The antibiotic regimen was changed midway through the Bair Hugger portion of the trial and then maintained constant for the rest of the study.
- The data was collected from only one hospital.

Neverthless, ECRI noted that the use of forced-air warming in orthopedic surgery was "particularly worrisome" and "especially concerning."

In response to the ECRI critique, an expert might conclude that the inclusion criteria chosen by ECRI for analyzing studies for this report were unreasonable. Of the 180 studies potentially related to the question, every single one of the studies was eliminated for one reason or another. When no studies met the criteria, the reasonableness of the criteria must be questioned.

The question of waste FAW heat causing PJIs will never be answered by a RCT. Given the low incidence of infection, a study would take several years and cost millions of dollars. 3M will certainly not sponsor an RCT because it will be almost impossible to show safety. The requirement to show "safety"—a negative, is much higher than the requirement to show "risk." Moreover, it is probable that given the available studies showing increased PJI risk from airborne contamination and the link to increased PJI rates with FAW, it would be unethical for an Institutional Review Board (IRB) to allow an RCT to have a control group of FAW-treated patients. Therefore, most likely there will never be an RCT done to show increased FAW risk, much less FAW safety.

ECRI's criticism regarding the antibiotic change is weak. The authors of the study show a graph of the raw infection data plotted over time. It is easy to eliminate that portion of the FAW patients that were treated with the first antibiotic and estimate that the remaining infection rates will be at least similar to the reported infection rates for each group. In fact, it appears that the infection rate for the FAW group after eliminating the first antibiotic group will most likely be higher than currently reported. Therefore, this is a specious argument.

The "single-hospital" criticism is also specious. Most published studies are done at a single hospital and are not "multi-center trials." Criticizing this study for being from a single center is unreasonable.

It is worth noting that similar infection reduction results to the McGovern study have been documented at Ridgeview Medical Center, Waconia, Minnesota. Surgeons there found that after discontinuing the use of FAW, their deep joint infection rate fell by 81% (1.55%→0.29%), 1,065 patients, 3 years. The 0.29% deep joint infection rate was achieved while using air-free conductive fabric warming.[2]

References:

1. Forced-air warming and surgical site infections, ECRI Institute. health Devices 2013;42-4:122-125.
2. Personal communication regarding information obtained from Robert Barnett MD, Orthopedic Surgeon, Ridgeview Medical Center, Waconia MN.

Conclusion

This litigation is ripe for the taking. Problems with the Bair Hugger were discovered some time ago, but it was only recently that enough peer reviewed literature became available for pursuing cases against Arizant/3M. While only one case has been filed to date, I foresee that many more will be filed in the future. The science is objectively sound and the theory is relatively easy to understand for future juries.

If you have questions regarding this guide or the litigation in general, please feel free to contact me.